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Evolution of the Acid-Base Status in Cardiac Arrest

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In a study of the evolution of acid-base status in 26 patients who had cardiopulmonary arrest in the operating room, it appeared that:

The determination of acid-base status within the first hour post-cardiac arrest is useful in differentiating final survivors from non-survivors.

Respiratory or combined acidosis carries a poor prognosis not evidenced for metabolic acidosis.

Late respiratory complications are more frequent in patients with initial combined acidosis.

Treatment should be instituted on the basis of frequent determinations of acid-base status, since accurate diagnosis of degree and type of acidosis cannot be done on clinical grounds only.

Recovery of consciousness is influenced by the type and severity of acidosis, less so by duration of arrest; and that high pCO_2 is associated frequently with unconsciousness after recovery of circulatory function.

SINCE THE INTRODUCTION and wide utilization of external and internal cardiopulmonary resuscitation techniques, there have been numerous reports on diverse factors related to their effectiveness. However, there are few reports concerning the acid-base balance in this emergency, and some of them give contradictory evidence regarding its possible prognostic value.

Our experience with 26 patients who had cardiopulmonary arrest while being submitted to surgical interventions in the Vargas Hospital of Caracas, and in whom treatment was begun without appreciable delay, will be presented here. We shall emphasize our findings on the acid-base balance and its relation to survival rates, cerebral damage, and duration of arrest, in order to establish whether there is any prognostic value in serial determinations of acid-base status besides their well-established value as a guide for treatment, and in the assessment of the actual condition of the patient.

Material and Methods

From 40 instances of cardiopulmonary arrest observed during three years in the Vargas Hospital and reported elsewhere,[1] 26 were selected to be reported here because serial determinations of arterial acid-base status had been carried out, beginning shortly after the diagnosis of arrest and then continuing for 24 to 48 hours.

Twenty-four of the accidents happened in the operating room and two in the cardiac catheterization laboratory. In both places, what with instrumental facilities, the presence of trained medical and paramedical personnel and the continuous monitoring, the arrest was recognized and treated without delay, and acid-base determinations were made within an hour of the beginning of arrest. In most cases, these determinations were made within ten minutes.

Cardiac arrest was defined as "a sudden and unexpected loss of pulse and arterial pressure"[2-4]; recovery was defined as "the conversion of ineffectual or absent cardiac action to effectual cardiac action, no longer requiring external or internal cardiac compression." The complete re-

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covery of neurological function was not applied to defining immediate success,[3] but its absence was taken as an indication of cerebral damage.

All 26 cases met the definition of temporary success of cardiopulmonary resuscitation, since all the patients were temporary survivors. In the rest of the report, the term *survivors* is applied to patients who left the hospital alive after a variable time of recovery.

The duration of arrest was computed as the duration of the resuscitation maneuvers, since the area in which the arrest occurred permitted diagnosis to be made without delay and the maneuvers were continued until recovery was achieved.

The acid-base data were obtained from 82 arterial blood specimens collected anaerobically at frequent and variable intervals shortly after the beginning of arrest, during the resuscitation maneuvers, and throughout the following 24 to 48 hours. Each specimen was analyzed immediately in duplicate, utilizing an Astrup AME 1-C (Radiometer) equipped with a glass electrode for pH determinations. Partial pressure of CO_2 (pCO_2) and base excess (BE) or base deficit (BD) were calculated, using the nomogram of Siggaard-Andersen, after the arterial blood was equilibrated with two different gas mixtures of known concentration.[5,6]

The classification of the acid-base alterations was based on the nomenclature accorded by the Ad Hoc committee of the New York Academy of Science in 1964.[7] Our normal values after correction for the hemoglobin concentration were: pH = 7.36-7.42, pCO_2 = 35-45 mm of mercury, and BE (base excess) +2 to −2 mEq per liter.

The management of the arrest was the responsibility of the medical team present at the time of the intervention. The method of treatment consisted of artificial ventilation via endotracheal intubation, external massage followed by internal compression when the first was not effective, or when the chest was already opened; electrical defibrillation when indicated, catecholamines (norepinephrine, isoproterenol), lidocaine, and other antiarrhythmic drugs; fluids and blood when indicated. Sodium bicarbonate was administered, utilizing as a guide the formula of Astrup and Mellemgaard[5] for calculation of the bicarbonate deficit corrected for the hemoglobin value and body weight. Usually, 50 mEq of 5 percent sodium bicarbonate was administered before the acid-base results were available but after the first specimen

TABLE 1.—*Clinical Summary of 26 Cases of Cardio-Pulmonary Arrest in the Operating Room*

Case No.	Age Sex	Procedure	ECG at beginning of arrest	Duration of arrest	Consciousness post-arrest	Outcome
1.	48F	Inguinal hernioplasty	V. fib.	15	Unconscious	Died—16 hr
2.	70F	Exploratory laparotomy	N.D.	15	Unconscious	*Died— 6 hr
3.	80F	Enucleation—right eye	Bradycardia, asystole	30	Conscious	Died—19 days
4.	33F	Left internal carotid artery aneurysm resection	N.D.	15	Unconscious	Died—1½ hr
5.	26M	Surgical correction of micrognathia	Asystole	18	Unconscious	Died—42 hr
6.	59F	Sigmoid colon polypectomy	V. fib.	7	Conscious	Died— 5 days
7.	14M	Cardiac catheterization	V. fib.	60	Conscious	Died—21 hr
8.	34F	Mitral valve commisurotomy	Asystole	10	Conscious	Died— 5 days
9.	16F	Exploratory laparotomy	N.D.	15	Unconscious	Died—31 hr
10.	85M	Partial gastrectomy	N.D.	65	Unconscious	Died—1½ hr
11.	56F	Abdomino-perineal resection	N.D.	9	Unconscious	Died—18 hr
12.	48F	Total thyroidectomy	Asystole	10	Unconscious	Died—30 hr
13.	67M	Femoro-popliteal bypass	Asystole	15	Unconscious	Died— 6 days
14.	60M	Partial gastrectomy	Asystole	55	Unconscious	Died— 2 hr
15.	30F	Hysterectomy	Asystole	50	Unconscious	Died— 2 hr
16.	28F	Hysterectomy	V. fib.	15	Unconscious	Died— 2 hr
17.	53M	Right radial neck dissection	N.D.	60	Unconscious	†Discharged—14 days
18.	78M	Abdominal aorta bypass graft	V. fib.	50	Conscious	Discharged—30 days
19.	25M	Brain tumor resection	N.D.	25	Unconscious	†Discharged—13 days
20.	68M	Cholecystectomy	N.D.	7	Conscious	‡Discharged—15 days
21.	67M	Right internal carotid artery aneurysm resection	N.D.	8	Conscious	‡Discharged—15 days
22.	8M	Cardiac catheterization	V. fib.	1	Conscious	Discharged—48 hr
23.	56F	Abdomino-perineal resection	Asystole	4	Conscious	Discharged—17 days
24.	60M	Aorto-iliac bypass	Asystole	7	Conscious	‡Discharged—30 days
25.	17M	Ulnar nerve neurorrhaphy	V. fib.	3	Conscious	‡Discharged—14 days
26.	55F	Abdomino-perineal resection	N.D.	5	Conscious	Discharged—30 days

Abbreviations: V. fib. = ventricular fibrillation; N.D. = not documented.

*Mild preop acidosis corrected. †Unconscious. ‡Returned to work.

was collected. Standard statistical techniques, such as contingency analysis, Student t test and linear correlation curves, were applied in order to assess the statistical significance of the data.

Results

Table 1 summarizes the clinical information on these patients. There was no difference in age or sex distribution between the survivor and the non-survivor groups. The overall age range was from 8 to 85 years, with a mean of 49 years. Half of the patients were females, but 11 of the 16 non-survivors were females and 8 of the 10 survivors were males.

Preoperative clinical and paraclinical tests showed 80 percent of the patients to have cardiovascular or pulmonary diseases, manifested as arteriosclerotic heart disease, rheumatic heart disease, chronic bronchitis, or emphysema.

Only six of the interventions were performed in emergency; all others were done as elective procedures. In two other instances, the arrest occurred while the patients were undergoing cardiac catheterization before a surgical procedure.

Of the 26 patients, 16 died after successful resuscitation and before being discharged from the hospital. Ten left the hospital alive and were considered survivors; however, two of these ten were transferred to a convalescent hospital because of severe neurological damage, without recovery of consciousness.

Evolution of acid-base status in survivors and non-survivors after cardiac arrest. In order to delineate differences in acid-base evolution, these patients were classified retrospectively into two groups. Group A consisted of ten patients who were discharged alive and considered as survivors; Group B consisted of 16 patients who died of complications resulting from their cardiac arrest before they were discharged.

Chart 1 shows a plotting of the acid-base results in these two groups, as taken sequentially during the first 48 hours post-arrest.

All 26 patients survived the first hour; specimens of blood taken during this period disclosed the "final" survivors to have normal or close to normal acid-base status (pH = 7.40 ± 0.03; CO_2 = 34.9 ± 3.8 mm of mercury; BE = 1.78 ± 3.1 mEq per liter). There was a statistically significant difference with the non-survivors, who had a severe combined acidosis (pH = 7.1 ± 0.08, pCO_2 = 63.2 ± 11.7, BE = −10.8 ± 2.7).

Six patients died during the following five hours.

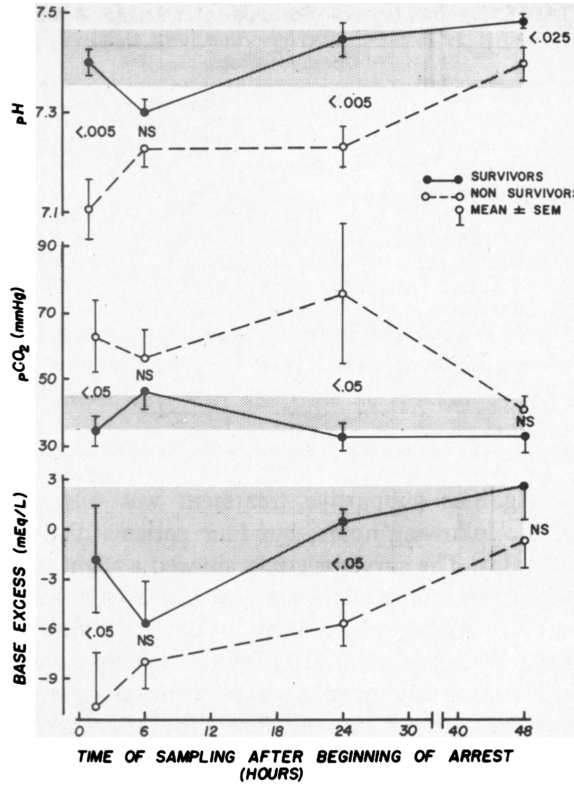


Chart 1.—Evolution of acid-base status in survivors and non-survivors after cardiac arrest. The non-survivor group showed a severe combined acidosis in the first sample post-arrest, and a further worsening of respiration in the next 18 hours. In contrast, the survivor group had a moderate metabolic acidosis initially, with fast restoration of normal acid-base status.

By this time, the survivor group had a moderate metabolic acidosis (pH = 7.31 ± 0.03, pCO_2 = 46 ± 5 mm of mercury; BE = −5.8 ± 2. mEq per liter), while the remaining non-survivors had improved their combined acidosis (pH = 7.23 ± 0.04; pCO_2 = 56.9 ± 8.4 mm of mercury; BE = −7.9 ± 1.6 mEq per liter).

During the next 18 hours, two more patients died. After readministration of bicarbonate, the survivors returned to a normal acid-base status (pH = 7.44 ± 0.04; pCO_2 = 32.2 ± 4 mm of mercury; BE = −0.8 ± 1.5 mEq per liter), showing a trend toward respiratory alkalosis due to slight hyperventilation. In the non-survivor group the metabolic derangement improved, as shown by a decrease in the base deficit, but many of them had a worsening of ventilation caused by previous gastric aspiration pneumonitis, pulmonary infections and atelectasis (pH = 7.22 ± 0.03, pCO_2 = 76 ± 21 mm of mercury; BE = −5.3 ± 2.1 mEq per liter). These differences in acid-base status were again statistically significant.

TABLE 2.—*Relation of Duration of Cardiac Arrest to Acid-Base Status,* Survival Rate, and Recovery of Consciousness*

	Duration of arrest	
	10 minutes or less	More than 10 minutes
No. of patients	11	15
pH	7.39±0.03	7.12±0.07†
pCO_2 mmHg	37.6±5.4	61.4±11.4‡
Base excess (BE) mEq/L	3.2±1.4	−10.8±2.8‡
Survival rate (percent)	63.6	20†
Recovery of consciousness (percent)	91	20†

Results are given as mean ± SEM.

*As determined in the first sample taken after cardiac arrest.
†$p<0.005$
‡$p<0.05$

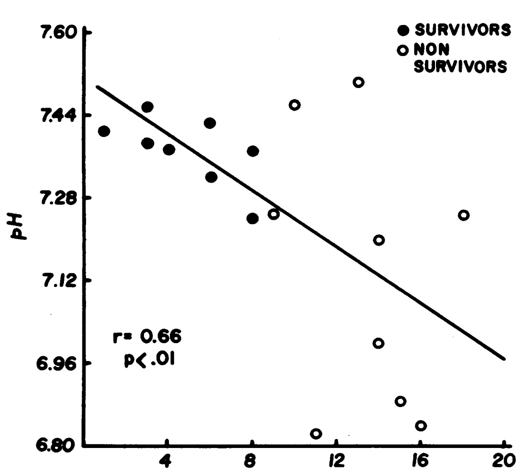
Vigorous supportive treatment was instituted in the following hours, but four patients died the next day. The survivor group showed a slight combined alkalosis at 48 hours post-arrest ($pH=7.48\pm0.01$, $pCO_2=33.2\pm4$ mm of mercury; BE $=2.5\pm0.6$ mEq per liter). The four remaining patients in the non-survivor group were returned to normal acid-base status for the first time after cardiac arrest ($pH=7.39\pm0.02$, $pCO_2=41.2\pm7$ mm of mercury; BE $=0.57\pm1.2$ mEq per liter). These patients died later of pulmonary complications subsequent to the cardiac arrest, two on the fifth day, one on the sixth day, and one on the nineteenth day.

Duration of arrest. In Table 2, the acid-base status determined during the first hour after cardiac arrest, the survival rate, and recovery of consciousness, are analyzed as a function of the duration of arrest, considering 10 minutes as an empirical discriminating time factor.

When an effective cardiac action was restored in less than 10 minutes, the acid-base status was slightly altered, survival rate was more than 60 percent and almost every patient regained consciousness. On the other hand, if arrest could not be reversed in that period of time, acid-base status showed a severe combined acidosis, survival rate was 20 percent, and only one out of every five patients regained consciousness. These differences were statistically significant ($p<0.05$).

An attempt was made in 18 patients to correlate their acid-base status at the end of the resuscitation maneuvers to the duration of arrest (Chart 2). Patients with alkalosis, or those in whom arrest lasted longer than 20 minutes, were excluded. A significant inverse correlation ($r=-0.66$, $p<0.02$) was obtained for pH and base excess, but it was not significant for pCO_2. This would indicate that the metabolic factor of the acidosis increases with longer periods of arrest, but the effect of treatment on these values precludes statement of any valid conclusion.

Recovery of consciousness. The status of consciousness was evaluated in every patient immediately after cardiovascular function resumed. As can be seen in Table 3, patients who remained unconscious after recovering from cardiac arrest had a severe combined acidosis in the specimens taken during the period of arrest, or in the first hour afterward ($pH=7.04\pm0.05$, $pCO_2=71.2\pm$ mm of mercury; BE $=-13.4\pm3$ mEq per liter).



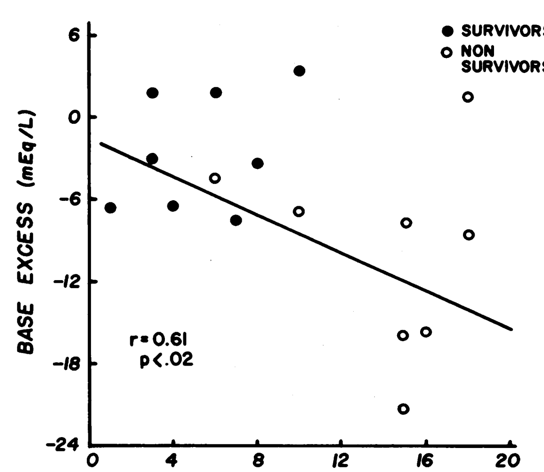


Chart 2.—Relationship of duration of arrest to pH and base excess values determined within one hour post-arrest. The metabolic factors of the acidosis post-cardiac arrest tend to increase significantly with longer duration of the arrest. The wide scattering of individual values indicates a high degree of variability due to influence of the treatment and the respiratory status on these parameters.

By retrospective analysis their survival rate was only 14.2 percent. In contrast, patients who regained consciousness had a normal acid-base status, coupled with a survival rate of 66.6 percent. Duration of arrest was longer in patients who did not regain consciousness (35 ± 12 minutes) than in those who did (15 ± 8 minutes)—($p > 0.01$). This difference was not statistically significant.

Type of acidosis. In the first blood specimen withdrawn, three patients showed normal acid-base status, three showed either respiratory (two) or metabolic (one) alkalosis, and the remaining 20 patients showed acidosis. Normal values were seen when the arrest lasted less than four minutes. The three instances of alkalosis were felt to be the consequence of either too vigorous pre-arrest or intra-arrest artifical ventilation, or an excessive amount of bicarbonate administered.

Of the 20 acidotic patients (Table 4), nine had predominant metabolic acidosis and 11 combined acidosis. A much poorer prognosis was associated with the presence of combined acidosis, since it was related to a survival rate of 11.1 percent. Patients with metabolic acidosis had a survival rate of 60 percent. All three patients with normal acid-base status were final survivors, but with alkalosis the outcome was variable.

TABLE 3.—*Relation of Recovery of Consciousness* to Acid-Base Status†*

	Conscious	*Unconscious*	*p*
pH	7.38 ± 0.04	7.04 ± 0.05	<0.001
pCO_2 mmHg	36.5 ± 5	71.2 ± 14	<0.05
BE mEq/L	-4.0 ± 2	-13.0 ± 3	<0.025
Survival rate (percent)	66.6	14.2	<0.005
Duration of arrest (minutes)	15 ± 8	35 ± 12	N.S.

Results are given as mean ± SEM

*As determined after recovery of cardiac function.
†Samples during the first hour post-arrest.

TABLE 4.—*Survival Rates in Acidosis Post-Cardiorespiratory Arrest*

Type of acidosis	*Survival rates*
Metabolic	60
Combined	11.1
p	<0.005

Discussion

One must distinguish cardiac arrest as a terminal event in a serious systemic disease from arrest occurring to a less seriously ill person during diagnostic (catheterization) or therapeutic (anesthesia, surgical operation) procedures. Most of the former will not survive arrest; most of the latter (40 to 90 percent) will recover initially if the precipitating factor causing the arrest can be removed. However, a very high proportion of these patients die shortly thereafter of complications of their cardiac arrest,[2-4,8,9] leaving only 5 to 30 percent as final survivors. In this group, then, it is essential to recognize prognostic indices which allow prevention of early and late mortality.

From our data, it is evident that the determination of acid-base status, when made within the first hour post-arrest, has prognostic value in delineating the survival possibilities of individual patients. The survivors had normal or close to normal acid-base status, as compared with a severe combined acidosis seen among the non-survivors, particularly in the presence of hypercapnia. These findings of severe acidosis in non-survivors after cardiac arrest have been reported previously.[10-12] Jude and coworkers[2] found significant differences in the severity of base deficit between survivors and non-survivors after cardiac arrest in the operating room. However, in contrast with our results, neither pH nor pCO_2 determined simultaneously were significantly different.

Subsequent specimens reflected the effects of the treatment. The slight metabolic acidosis observed in the survivors during the six hours after arrest can be explained by increased levels of lactic acid and other acidotic metabolities washed out from ischemic peripheral capillary beds, once circulation is re-established.[13] The CO_2 produced by the buffering action of the blood might have induced a slight increase in pCO_2. Since in these patients there was little deviation from the normal A-B pattern in the first speciman, bicarbonate was not administered in large amounts, allowing the acidosis to be evident. At this stage, the appearance of metabolic acidosis did not alter prognosis. On the other hand, large amounts of bicarbonate and appropriate assisted ventilation were given accordingly to the non-survivors, thus substantially improving their acid-base balance.

Before the end of the first 24 hours post-arrest, there was a worsening of ventilatory function in many non-survivors, although they were maintained under positive-pressure artificial ventilation and close surveillance. This worsening was the result of aspiration of gastric fluids, atelectasis, broncho-pneumonias, and sudden interruption of artificial ventilation due to dislodgement or replacement of intratracheal tubes.

It is important to note that patients with high pCO_2 at the periarrest period were more prone to those respiratory complications, which were frequently fatal. By contrast, survivors returned to normal A-B status during the first 24 hours.

As is well established in previous reports,[2,4,14,15] the duration of arrest influenced prognosis. Patients whose arrest lasted less than 10 minutes had 60 percent survival rate, as compared with less than 20 percent when arrest lasted longer than 10 minutes. The longer duration of arrest was accompanied by severe combined acidosis and late recovery of consciousness, as could be expected from the more severe degree of tissue hypoxia and cerebral edema.

A good correlation between buildup of metabolic acidosis and duration of arrest has been demonstrated experimentally[16] and also reported in some clinical studies.[17] This correlation permitted the development of formulae for calculation of bicarbonate dosage, based on duration of arrest and body weight. In our series, although there was a significant degree of correlation between duration of arrest and pH or base deficit (Chart 2), it was not accurate enough for development and application of such formulae. The status of ventilation before and during arrest modified the effects of duration of arrest on the acid-base status, precluding the application of formulae not based on the actual pH, pCO_2 and BE of blood for administration of bicarbonate.

Regarding predisposing factors, Camarata et al[8] recently found acidosis preceding cardiac arrest in a high proportion of critically ill patients. When no preceding acidosis was present, an acute anoxic period was directly related to the arrest. Our patients did not have a critical condition as a cause of acidosis, but the large incidence of combined acidosis in the first speciman suggests that hypoxemia and hypercapnia during the surgical procedure were major causes of arrest, and determinants of late survival rate.

The recovery of consciousness, as mentioned before, was late, if ever present, in the acidotic patients, who also had longer duration of arrest and poor survival rate (Table 3). Particularly high pCO_2 was related to long periods of unconsciousness and cerebral damage. Hypoxia and hypercapnia cause cerebral vasodilation and subsequent cerebral edema[14,18] expressed clinically as unconsciousness. The fact that arrest lasted longer, although it was not statistically significant in unconscious patients, suggests that other variables such as the degree of acidosis and pCO_2 retention were greater influences on the recovery of consciousness.

We observed a very low survival rate in cases of combined acidosis (11 percent) as compared with cases of metabolic acidosis (60 percent), a fact which corroborates the findings of Chazan el al[11] and gives this early finding an ominous prognostic value after cardiac arrest.

Some observations regarding treatment are pertinent. Since it was not possible to predict the type and severity of acidosis on clinical grounds, no formula for administration of bicarbonate based solely on duration of arrest and body weight could be developed. Thus, the actual determination of the acid-base status is essential for guiding and evaluating therapy.[2,11,14]

Ventilatory failure was frequently associated with death; therefore, every effort should be aimed at ensuring good ventilation as early as possible. This will also facilitate the treatment of metabolic acidosis in such cases by eliminating the excess of CO_2 produced by the buffering action of bicarbonate, and improving oxygenation.[12,13] Close surveillance and preventive measures must be taken to avoid late respiratory failure.

REFERENCES

1. Oletta JF, Carrasco HA, Plaza J, et al: Reanimacion cardiorespiratoria. Bol Soc Venez Cir 24:127-136, Feb 1970
2. Jude JR, Boolook H, Nagel E: Cardiac resuscitation in the operating room—Current status. Ann Surg 171:948-955, Jun 1970
3. Peschin A, Coakly CS: A three year review of 734 cardiopulmonary arrests. South Med J 63:506-510, May 1970
4. Jeresaty RM, Goday TJ, Liss JP: External cardiac resuscitation in a community hospital. Arch Int Med 124:588-592, Nov 1969
5. Siggaard-Andersen O: The Acid Base Status of the Blood. Williams & Wilkins Co., 2nd ed Baltimore, 1964
6. Siggaard-Andersen O, Engel KA: New acid-base nomogram—An improved method for the calculation of the relevant blood acid-base data. Scand J Clin Lab Invest 12:3, 311, 1960
7. Ad-Hoc committee of the New York Academy of Science. Statement on acid-base terminology. Ann Int Med 65:885-890, Nov 1965
8. Camarata S, Weil MH, Hanashiro PK, et al: Cardiac arrest in the critically ill—1. A study of predisposing causes in 132 patients. Circulation 44:688-695. Oct 1971
9. Hollingsworth JH: The results of cardiopulmonary resuscitation—A 3 year university hospital experience. Ann Int Med 71:459, Sep 1969
10. Edmonds-Seal J: Acid-base studies after cardiac arrest. Acta Anaesth Scand 10: suppl 23:235-241, Aug 1966
11. Chazan JA, Stenson R, Kurland GS: The acidosis of cardiac arrest. N Engl J Med 278:360, Feb 1968
12. Kirby BJ: Cardiac resuscitation—Clinical and biochemical studies. Postgrad Med 43:75-80, Feb 1967
13. Fillmore SJ, Shapiro M, Killip T: Serial blood gas studies during cardiopulmonary resuscitation. Ann Int Med 72:465 Apr 1970
14. Gilston A: Clinical and biochemical aspects of cardiac resuscitation. Lancet 2:1039, Nov 1965
15. Harley HRS: Reflections on cardiopulmonary resuscitation. Lancet II:1-4, Jul 1966
16. Ledigham I, Norman JN: Acid-base studies in experimental circulatory arrest. Lancet II:967, Nov 1962
17. Stewart JSS: Management of cardiac arrest, with special reference to metabolic acidosis. Br Med J 1:476-478, Feb 1964
18. Malm OJ: Treatment of acidosis and electrolyte disturbances in asphyxia and cardiac arrest. Acta Anaesth Scand 12: suppl. 29:165-182, May 1968